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# COMPLICATIONS OF SUBARACHNOID HEMORRHAGE ON THE HEART

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Review began 23/09/2024
Review ended 20/10/2024
Published 31/10/2024



DOI: https://doi.org/10.5281/zenodo.14039003

## Abstract

**This is the first time the medical literature has discussed a teenager's case of subarachnoid hemorrhage leading to ventricular tachycardia. This is supported by previous researchers which have been carried out together with their data showing that a functional pathway from the brain to the heart exists. This article also elaborates on the early diagnosis of subarachnoid hemorrhage and its related cardiac abnormalities, together with the importance of Holter monitoring in this group of patients.**

**Categories:** Cardiology
**Keywords:** Subarachnoid hemorrhage, ventricular tachycardia, cardiac abnormalities, holter monitoring

## Introduction

Cardiac abnormalities are present in more than 50% of patients with subarachnoid hemorrhage where most of them are at high risk of developing severe ventricular arrhythmias, malignant arrhythmias and are also responsible for sudden deaths in some circumstances. Data have showed that delays in diagnosis and treatment of subarachnoid hemorrhage increase the risk of death by close to 400%. The purpose of this article is to bring awareness about the complications of subarachnoid hemorrhage on the heart.

## Case Report

A 15 years old female patient was brought to the emergency department. She was comatose with a glasco coma scale of 3 and had 4 episodes of projectile vomiting prior to coming to hospital. She was immediately intubated. Her parents denied having prior symptoms, history of fall or head trauma. She does not have any past medical, surgical, allergic, or significant family history. The initial 12 leads electrocardiogram (ECG) showed monomorphic ventricular tachycardia Figure.1 with a heart rate of 150 bpm and a BP of 90/60 mmHg. She underwent successful direct current cardioversion to sinus rhythm on the third time Figure 2. IV Infusion amiodarone 450mg was started for the next 12 hours and was admitted to the intensive care unit. CT scan brain showed generalized edema, acute cerebral bleeding extending into the 3rd and 4th ventricles and right tentorium cerebelli. An external ventricular drain was inserted. Unfortunately, the patient was in a critical clinical condition and died after 72 hours.

## Discussion

What killed the patient? Cerebral hemorrhage or ventricular tachycardia?

Patients presenting with a subarachnoid hemorrhage-SAH frequently demonstrate ECG changes [1]. Cardiac manifestations are a crucial hint to help in early diagnosis and prognosis stratification of cerebral damage. ECG changes and arrhythmias can be noticed during a patient's initial presentation [3]. Hence, it is important to identify the physical and diagnostic findings that may correlate with subarachnoid hemorrhage.

One may wonder whether cardiac arrhythmias are secondary to severe cerebral damage or contrarily are independent aspects which may drive to this prognosis. Many studies have shown that SAH may secondarily cause myocardial damage and cardiac arrhythmias. Several authors have described hypothalamus lesions secondary to increased intracranial pressure from SAH is responsible for the production of arrhythmias in SAH patients.

In a study done by Andreoli et al, out of 70 patients with SAH, 64 had arrhythmias (91%), 29 had severe cardiac arrhythmias (41%) and 3 had malignant ventricular arrhythmias that is ventricular flutter and torsade de pointe with no significant difference between sexes.[5]

In another study of 135 stroke patients, 61% had cardiac arrhythmias during the first 3 days after the stroke. Since 69% of them were known cardiac patients, most of the arrhythmias were due to pre-existing cardiac complications. However, despite having a high number of cardiac patients in this research, this seems unconvincing to explain such a high rate of arrhythmias. [5]

Moreover, other medical articles also showed ST segment elevation and depression are quite common in cerebral damage patients where they had raised troponin with unremarkable coronary angiography which is due to high level of circulating catecholamines. [2, 3] So far, all reported cases in the scientific literature were adults. However, we firmly believe that this is the first time a teenager's case report is being discussed regarding this matter.

To support the above literature, many scientific journals described the below. Animals were used as models to demonstrate that cardiac arrhythmias can be caused secondarily due to intracranial hemorrhage whereas cardiac arrhythmias in human beings were incidental findings. Misra and Prasad injected 5ml of blood into the orbitofrontal region of dogs' brain; 100% of them had sinus irregularity where 57% had cardiac extrasystoles as a result of this lesion. Furthermore, Mauck, Hockman and Hoff published that electrical stimulation of the central nervous system-CNS centers produced arrhythmias in animals. Ventricular tachycardia and ventricular fibrillation were observed in monkeys while premature ventricular complexes and atrioventricular block were produced in cats. Weinberg and Foster observed that stimulation of the lateral and posterior hypothalamus produced T waves changes, bigeminy, trigeminy, ventricular tachycardia and WPW syndrome. Another study showed that electrical stimulation of the vagus nerve induced cardiac arrhythmias in animals. Simultaneous stimulations of both nerves demonstrated the same spectrum of cardiac abnormalities which were obtained with stimulation of the midbrain and posterior hypothalamus. However, these were abolished with bilateral vagotomy.

Connor brought forward that myocardial damage after cerebrovascular accident was the result of a stimulation of the autonomic nervous system and an elevation in the level of circulating catecholamines due to the activated sympathetic centers in the hypothalamus. These are enough evidences which showed that functional pathways from the brain to the heart exist. [7, 8]

## Conclusion

Considerable evidence exists to supports that SAH may secondarily cause myocardial damage and life-threatening cardiac arrhythmias in patients followed by death. We believe that the cause of cerebral bleeding in this child could have been a ruptured aneurysm. Therefore, to have a better prognosis, intracranial hemorrhage should be drained as soon as possible to decrease intracranial pressure thus minimizing cardiac complications and mortality [6]. In addition, delays in diagnosis and treatment increase the risk of death by close to 400% [1]

Moreover, holter monitoring should be initiated within 48 hours in all patients with cerebral hemorrhage to evaluate the frequency, time, type and severity of cardiac arrhythmias if any and to diagnose the cardiac rhythm disorders early because these patients are at higher risk of developing arrhythmias in the acute phase of SAH. Cardiac arrhythmias should be considered as a highly probable cause of death in this group of patients.

## Conflict of interest :

None declared

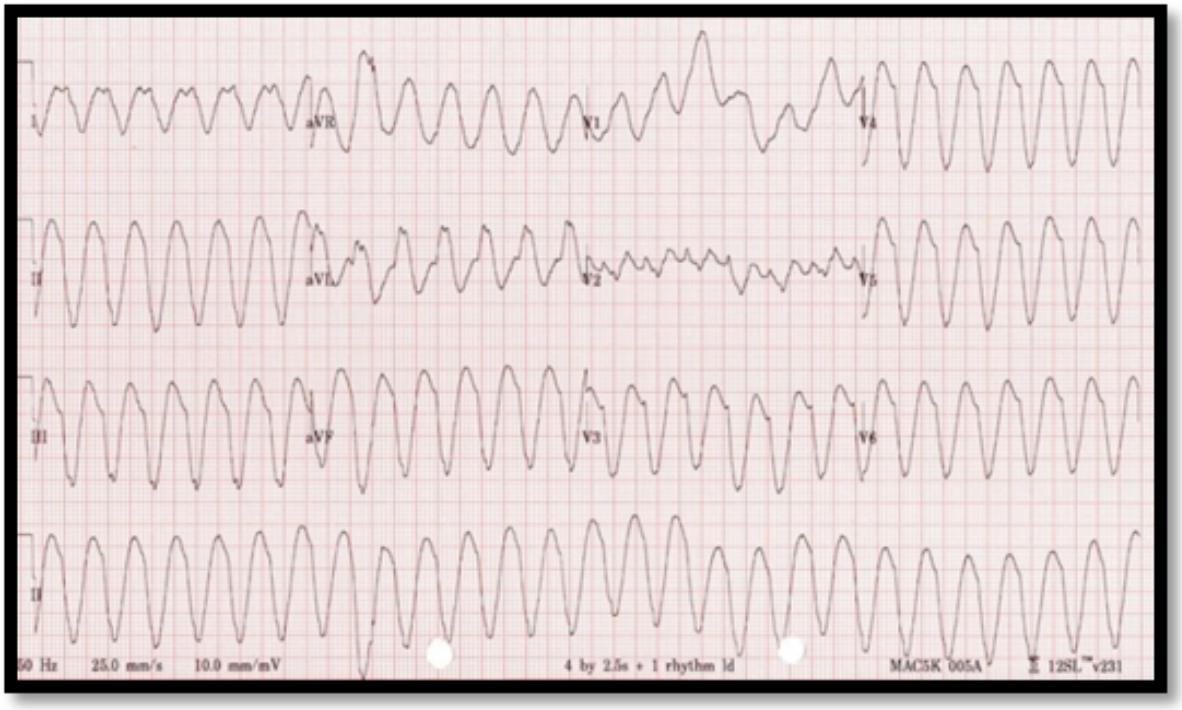


**Figure 1**

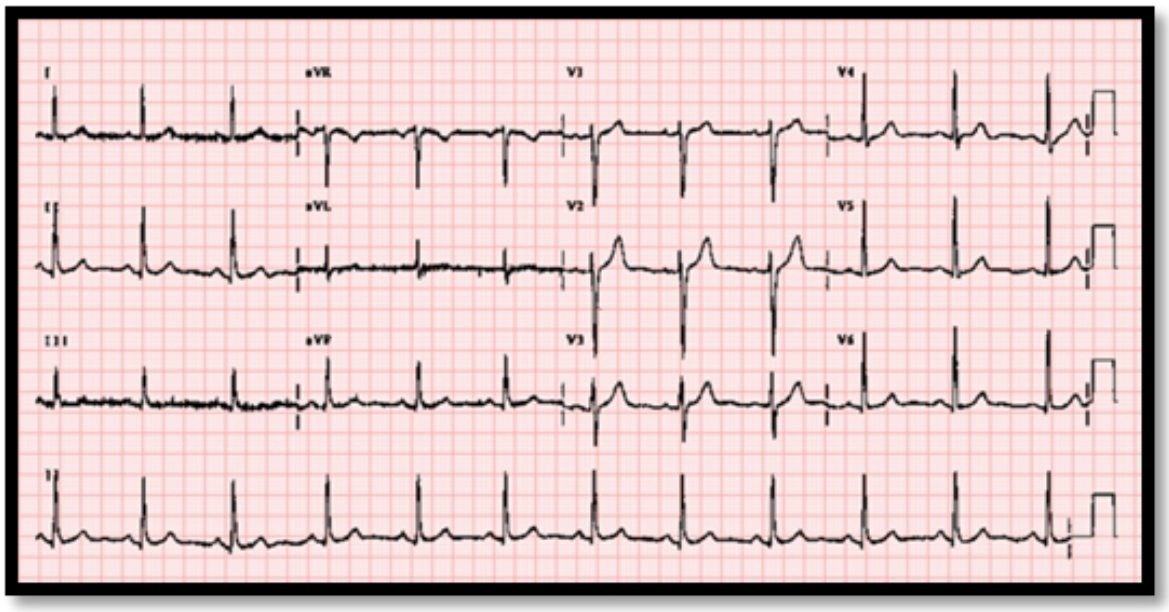


**Figure 2**

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